JACC Vol. 15, No. 2
February 1990:122A

AORTIC VALVE MORPHOLOGY: AN IMPORTANT DETERMINANT OF PROXIMAL REGURGITANT JET WIDTH BY DOPPLER COLOR MAPPING

Anne L. Taylor, M.D., F.A.C.C., Eric Eichhorn, M.D., F.A.C.C., Elizabeth M. Brickner, M.D., Robert C. Eberhart, Ph.D., Paul A. Grayburn, M.D., F.A.C.C. UT Southwestern, Dallas, TX.

In vitro and in vivo studies suggest that proximal aortic regurgitant jet width (RW) predicts severity of aortic regurgitation (AR). The influence of aortic valve morphology (AVM) on RW has not been studied. Hypothesis: despite equal cross-sectional area, differences in AVM may influence RW and thus, estimates of severity of AR. Methods: AVM's simulating degenerative, rheumatic, bicuspid and circular valve orifices in two cross-sectional areas (0.2 cm^2 and 0.7 cm^2) were placed in a flow model using two initial gradients (50 and 100 mmHg) to produce simulated AR jets. Doppler color flow maps (CFM's) were obtained from parasternal (P) and apical (A) positions with gain, FPS, low velocity reject, and depth held constant. The mean of three RW's for each valve shape, in each orifice area and at each initial gradient were compared by three factor ANOVA. Results: Aortic valve morphology produced significant variation in RW for both valve areas in P and A views (p=0.0001 by ANOVA). Bicuspid shapes in both areas yielded RW's significantly different from all other shapes. Valve area also significantly influenced proximal regurgitant jet width (p=0.0001) in both views, while initial pressure gradient was less important. Conclusions: In an in vitro flow model, AVM introduced significant variability (which was independent of orifice area) in the measurement of proximal RW by CFM. Estimates of severity of aortic regurgitation may therefore be importantly influenced by aortic valve morphology.

ELECTRICAL ALTERNANS AS A PRECURSOR TO VENTRICULAR ARRHYTHMIAS: A CELLULAR MECHANISM.

David S. Rosenbaum, M.D., Jeremy N. Ruskin, M.D., F.A.C.C., Lance Jackson, B.S., Richard J. Cohen M.D., Ph.D., Guy Salama, PhD.. Harvard Medical School and M.I.T., Boston, MA.

Though electrical alternans (EA) of the surface ECG has been associated with clinical and experimental ventricular arrhythmias, the mechanism of increased arrhythmia susceptibility has remained obscure. We examined the electrophysiologic properties of the myocardial syncytium during EA utilizing high resolution optical mapping with voltage sensitive dyes. Action potentials (AP) were recorded simultaneously from 124 sites in a 1x1 cm region of the left ventricular surface of 10 Langendorff guinea pig hearts. While stimulating (300 bpm) the center of the optical field, Surface ECGs and AP's were measured during perfusion with oxygenated Ringers (control) and after 30 minutes of either hypoxia or hypothermia (25°C). ECGs and APs acquired during 50 consecutive even beats were signal averaged and compared to signal averaged data obtained during 50 consecutive odd beats. Significant EA of surface ECG morphology (EA_{ECG}) was elicited by both hypoxia and hypothermia and was attributable to beat-to-beat alternation of AP shape (EA_{AP}) and duration (EA_{APD}).

*p<0.02	Control	Hypoxia	Hypothermia
EA_{ECG} (%)	4.3 ±4.1	24.5 ±9.1*	61.1 ±43.0*
EA_{AP} (%)	1.3 ±0.6	7.6 ±4.2*	41.4 ±16.2*
EA_{APD} (%)	0.4 ±0.6	1.5 ±0.8*	10.0 ±1.6*

There was no evidence for alternating activation sequence, conduction velocity, or AP maximum upstroke velocity. EA_{AP} primarily involved alternating attenuation of phase 2, and to a lesser extent phase 3 of the AP which gave rise to EA_{APD}. There was significant spatial inhomogeneity of EA_{AP} as cells with longer APD's tended to oscillate most. This resulted in accentuated spatial dispersion of repolarization within injured myocardium compared to controls. The spatial variability of EA_{AP} was not related to pacing site or fiber orientation.
CONCLUSIONS: These data demonstrate that EA as manifested on the surface ECG is not due to alternating wavefront propagation or wavefront fractionation but rather due to EA_{AP}. Enhanced spatial dispersion of recovery owing to EA_{APD} may explain the pathophysiologic coupling of EA to arrhythmogenesis.

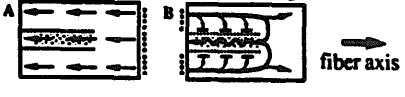
Tuesday, March 20, 1990
4:00PM–5:30PM, Room 41
Electrophysiologic Substitute of Tachyarrhythmias

ELECTROTONIC DRAG IN A CONDUCTING ISTHMUS WITH RESISTIVE BARRIERS: IMPLICATIONS FOR SLOW CONDUCTION IN NON-UNIFORMLY ANISOTROPIC MYOCARDIUM.

Michael D. Lesh, MD, Martin Pring, PhD, Joseph F. Spear, PhD, FACC. University of California San Francisco, San Francisco, CA

Myocardial infarction, by superimposing poor cell-to-cell coupling on normal fiber geometry, results in non-uniform anisotropy. We hypothesized that in addition to the *direct* slowing caused by poor coupling, electrotonic current *passing through* lines of block can slow conduction in a directionally-dependent fashion. We tested this in a computer model simulating a 6x4mm sheet of ventricular myocardium as a grid of 2400 elements obeying Beeler-Reuter(BR) ionic-current kinetics (see figure below). BR parameters were uniform throughout. Longitudinal and transverse resistivity (R) were 2.5 and 10KΩcm respectively. Dotted lines in the figure indicate barriers of poor coupling which delineate a conducting isthmus(stippled area). Barrier R was varied from 20KΩcm to ∞. For each, we performed 2 types of experiments (figure). A: pacing(•) an entire edge; B: pacing external to the isthmus to produce a figure-of-eight conduction pattern. In type A expts. conduction was uniform(arrows) with velocity (θ) =19cm/s and $\dot{V}_{max}$=105V/s. In type B experiments, whenever barrier R was greater than 25KΩcm the impulse blocked at the barriers and proceeded in opposite directions outside and inside the isthmus. *Outside* the isthmus, θ and $\dot{V}_{max}$ were uniform as they had been in A. *Inside*, heterogeneous slowing was noted, the degree of which depended on barrier R and spacing. For example, in the center of the isthmus when barrier R was 25KΩcm and spacing was 500μm, θ=6cm/s and $\dot{V}_{max}$=10V/s (68% and 90% reduction from baseline). When R=∞(complete uncoupling), θ and $\dot{V}_{max}$ were again normal as in A.

We concluded that in figure-of-eight as opposed to uniform activation, $\dot{V}_{max}$ was reduced and conduction slower because electrotonic current *passing through* lines of block resulted in a prolonged depolarization, inactivating Na channels and presenting the impulse passing *around* the barriers with reduced excitability. The magnitude of electrotonus, and therefore of slowing, depended on barrier R and spacing. "Electrotonic drag" in non-uniformly anisotropic myocardium may contribute to conduction slowing during reentry involving a narrow central isthmus.



PRECISION OF PACE MAPPING IN DETECTING THE ORIGIN OF CANINE SUSTAINED MONOMORPHIC VENTRICULAR TACHYCARDIA

Jiunn-Lee Lin, MD, David J Wilber, MD, Duke Du, MS, David S Rosenbaum, MD, Jeremy N Ruskin, MD,FACC, Hasan Garan, MD FACC, Massachusetts General Hospital, Boston, MA

To define the precision of ventricular pace-mapping (PM) in predicting the site of earliest activation (SEA) during sustained monomorphic ventricular tachycardia (SMVT), we quantitated the difference in surface ECG waveform between SMVT and PM by calculating a metric representing their combined vectorial difference defined by $\Delta = \left[(X_i-X_j)^2 + (Y_i-Y_j)^2 + (Z_i-Z_j)^2\right]^{\frac{1}{2}}$ (X,Y,Z, orthogonal lead components, i=VT, j=PM). In a canine model of myocardial infarction, 41 distinct SMVTs were induced by programmed stimulation in 10 animals and a unique SEA was defined for each SMVT by activation sequence mapping with bipolar recordings from a standardized array of 60 endocardial and epicardial ventricular sites. PM from the same 60 sites was performed at cycle lengths comparable to those of SMVTs. The orthogonal surface ECG waveforms generated from each of 60 pacing sites were analyzed using a computerized template-matching algorithm and compared to orthogonal surface SMVT waveforms using Δ (60 comparisons for each SMVT). For each SMVT the PM site which yielded the best match by minimizing Δ was chosen as the predicted site and was compared to the SEA determined from local activation times assigned during SMVT while blinded to the site predicted by Δ. In 35 (85%) of the 41 SMVTs, these two sites were either identical (19 SMVTs) or lay within 1 cm of each other (16 SMVTs), whereas in the remaining 6 SMVTs the two sites were disparate (>1 cm apart). Considering the temporal and spatial resolution of activation sequence mapping, we conclude that this metric can predict the SEA of SMVT with excellent resolution (within 1 cm) in 85% of the tachycardias.